What is claimed is:

1. An antagonist that inhibits or an agonist that activates an activity a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Ki(app);
competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of Kmapp for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length;
feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.

2. A method for the treatment of an individual having need to inhibit or activate Fab I polypeptide comprising the steps of: administering to the individual a antibacterially

effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Kiapp);
competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of Kmapp for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length;
feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.

3. A method for the treatment of an individual infected with a bacteria comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates an activity of a polypeptide selected from

the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Ki(app));
competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of Kmapp for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.

4. The method of claim 3 wherein said bacteria is selected from the group consisting of a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.

5. A method for the treatment of an individual having need to inhibit or activate Fab I polypeptide comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agoinist that activates an activity of Fab I selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Kiapp);
competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of Kmapp for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.

6. A method for the treatment of an individual infected with a bacteria comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or an agonist that activates that activates an activity of Fab I selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Ki(app));
competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of Kmapp for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.

7. The method of claim 6 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus, Staphylococcus aureus,* a member of the genus *Streptococcus, and Streptococcus pneumoniae.*

8. A method for the treatment of an individual infected by *Streptococcus pneumoniae* comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits or anagonist that activates an activity of *Streptococcus pneumoniae* Fab I selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Ki(app));

competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of Kmapp for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.

9. An antagonist that inhibits an activity of a polypeptide selected from the group consisting of: a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, , wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Ki(app));
competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;

binding of NADH and NADPH by FabI;

oxidation of NADH and NADPH by FabI;

ratio of Kmapp for NADH as compared to NADPH;

use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

sequential binding of NADH and crotonoyl CoA by Fab I;

increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;

binding of multiple palmitoyl CoA molecules to Fab I;

negative cooperativity in the binding of CCA;

formation of an dimeric quaternary structure;

formation of an tetrameric quaternary structure;

formation of an oligomeric quaternary structure;

binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and

NADH binding to Fab I prior to or simultaneous with ACP binding.

10. A method for the treatment of an individual having need to inhibit Fab I polypeptide comprising the steps of administering to the individual a antibacterially effective amount of an antagonist that inhibits an activity of a polypeptide selected from the group consisting of a polypeptide comprising an amino acid sequence which is at least 90% identical to the amino acid sequence of SEQ ID NO:2 or 4, and a polypeptide comprising an amino acid sequence as set forth in SEQ ID NO:2 or 4, . wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Ki(app);

competitive inhibition by Apo-ACP versus crotonoyl CoA;

induction of negative cooperativity with respect to CCA binding;

use of NADH and NADPH as substrates by Fab I;

binding of NADH and NADPH by FabI;

oxidation of NADH and NADPH by FabI;

ratio of Kmapp for NADH as compared to NADPH;

use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

sequential binding of NADH and crotonoyl CoA by Fab I;

increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;

binding of multiple palmitoyl CoA molecules to Fab I;

negative cooperativity in the binding of CCA;

formation of an dimeric quaternary structure;

formation of an tetrameric quaternary structure;

formation of an oligomeric quaternary structure;

binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and

NADH binding to Fab I prior to or simultaneous with ACP binding.

11. A method for inhibiting an activity of Fab I polypeptide comprising the steps of contacting a composition comprising said polypeptide with an effective amount of an antagonist that inhibits an activbity of Fab I, wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Kiapp);

competitive inhibition by Apo-ACP versus crotonoyl CoA;

induction of negative cooperativity with respect to CCA binding;

use of NADH and NADPH as substrates by Fab I;

binding of NADH and NADPH by FabI;

oxidation of NADH and NADPH by FabI;

ratio of Kmapp for NADH as compared to NADPH;

use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;
binding of multiple palmitoyl CoA molecules to Fab I;
negative cooperativity in the binding of CCA;
formation of an dimeric quaternary structure;
formation of an tetrameric quaternary structure;
formation of an oligomeric quaternary structure;
binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and
NADH binding to Fab I prior to or simultaneous with ACP binding.

12. A method for inhibiting an activity of Fab I, wherein said activity is selected from the group consisting of:
uncompetitive inhibition by Apo-ACP versus NADH (Ki(app);
competitive inhibition by Apo-ACP versus crotonoyl CoA;
induction of negative cooperativity with respect to CCA binding;
use of NADH and NADPH as substrates by Fab I;
binding of NADH and NADPH by FabI;
oxidation of NADH and NADPH by FabI;
ratio of Kmapp for NADH as compared to NADPH;
use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;
sequential binding of NADH and crotonoyl CoA by Fab I;
increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;
competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;

binding of multiple palmitoyl CoA molecules to Fab I;

negative cooperativity in the binding of CCA;

formation of an dimeric quaternary structure;

formation of an tetrameric quaternary structure;

formation of an oligomeric quaternary structure;

binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and

NADH binding to Fab I prior to or simultaneous with ACP binding.

13. The method of claim 12 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.

14. A method for inhibiting a growth of bacteria comprising the steps of contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Kiapp);

competitive inhibition by Apo-ACP versus crotonoyl CoA;

induction of negative cooperativity with respect to CCA binding;

use of NADH and NADPH as substrates by Fab I;

binding of NADH and NADPH by FabI;

oxidation of NADH and NADPH by FabI;

ratio of Kmapp for NADH as compared to NADPH;

use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

sequential binding of NADH and crotonoyl CoA by Fab I;

increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;

binding of multiple palmitoyl CoA molecules to Fab I;

negative cooperativity in the binding of CCA;

formation of an dimeric quaternary structure;

formation of an tetrameric quaternary structure;

formation of an oligomeric quaternary structure;

binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and

NADH binding to Fab I prior to or simultaneous with ACP binding.

15. The method of claim 14 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.

16. A method for inhibiting a Fab I polypeptide comprising the steps of contacting a composition comprising bacteria with an antibacterially effective amount of an antagonist that inhibits an activity of Fab I, wherein said activity is selected from the group consisting of:

uncompetitive inhibition by Apo-ACP versus NADH (Ki(app);

competitive inhibition by Apo-ACP versus crotonoyl CoA;

induction of negative cooperativity with respect to CCA binding;

use of NADH and NADPH as substrates by Fab I;

binding of NADH and NADPH by FabI;

oxidation of NADH and NADPH by FabI;

ratio of Kmapp for NADH as compared to NADPH;

use of NADH and crotonoyl CoA as substrates by Fab I in a sequential kinetic mechanism;

sequential binding of NADH and crotonoyl CoA by Fab I;

increasing inhibition of FabI by saturated fatty acyl CoA's of increasing chain length; feedback regulatory mechanism of Fab I by saturated fatty acyl CoA's;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA;

competitive inhibition by palmitoyl CoA versus crotonoyl CoA modulation through binding of multiple palmitoyl CoA molecules to Fab I;

binding of multiple palmitoyl CoA molecules to Fab I;

negative cooperativity in the binding of CCA;

formation of an dimeric quaternary structure;

formation of an tetrameric quaternary structure;

formation of an oligomeric quaternary structure;

binding of Fab I by pseudo-product inhibitors beta-NADP+ or palmitoyl coA; and

NADH binding to Fab I prior to or simultaneous with ACP binding.

17. The method of claim 16 wherein said bacteria is selected from the group consisting of: a member of the genus *Staphylococcus*, *Staphylococcus aureus*, a member of the genus *Streptococcus*, and *Streptococcus pneumoniae*.